Objective: This study was undertaken to investigate the potential role of Irbesartan in amelioration of myocardial I/R injury induced by ligation of coronary artery in a rat model.
Materials & Methods: Adult male Swiss Albino rats were randomized into 4 equal groups. Group (1) sham group: rats underwent the same anesthetic and surgical procedure as the control group except ligation of LAD coronary artery, Group (2) control group: rats subjected to regional ischemia for 25 min and reperfusion for 2 hours by ligation of LAD coronary artery, Group (3) control vehicle group: rats received vehicle of Irbesartan (normal saline) via I.P injection and subjected to regional ischemia for 25 min and reperfusion for 2 hours by ligation of LAD coronary artery, Group (4) Irbesartan treated group:rats pretreated with Irbesartan 3mg/kg i.p. 30 minutes before ligation of LAD coronary artery. At the end of experiment (2 hr of reperfusion), blood samples were collected from the heart for measurement of plasma level of cardiac troponin I(cTn I). after that the heart was harvested and divided in to 3 parts, one part was used for measurement of SSDNA (a marker for apoptosis) and another part was homogenized for the measurement of tissue tumor necrosis factor-α (TNF-α) and interleukin-1β (IL-1β) and interleukin-6,Monocyte chemo attractant protein-1 and Macrophage inflammatory protein -1α and the last part for histopathology study.
Results: Compared with the sham group, levels of myocardial TNF-α & IL-1β, IL-6, MCP-1. MIP-1alpha; plasma cTn I were increased ($p<0.05$). Histologically,all rats in control group showed significant ($p<0.05$) cardiac injury. Furthermore all rats in control group showed significant ($p<0.05$) apoptosis. Irbesartan significantly counteract the increase in myocardium level of TNF-α, IL-1B, IL-6, MCP-1, MIP-1alpha, plasma cTnI& SSDNA apoptotic marker ($p<0.05$). Histological analysis revealed that Irbesartan markedly reduced ($p<0.05$) the severity of heart injury in the rats underwent LAD ligation procedure.
Conclusion: The results of the present study reveal that Irbesartan may ameliorate myocardial I/R injury in rats via interfering with inflammatory reactions & apoptosis which induced by I/R injury.

OP-131

Phenolic Compounds Ameliorates Doxorubicin Induced Cardiac Injury Via Interfering with Inflammatory and Oxidative Pathways

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Background: The importance of doxorubicin, as a potent antitumor antibiotic, is limited by the development of life-threatening cardiomyopathy.. It has been shown that free radicals are involved in doxorubicin-induced toxicity. Doxorubicin causes the generation of free radicals and the induction of oxidative stress, associated with cellular injury. Phenolic compounds are a chemical family whose members have one or more hydroxyl groups attached directly to an aromatic ring. phenolic compounds can frequently act as free-radical scavengers.
Objective: This study was undertaken to investigate the cardioprotective potential of some phenolic compounds namely caffeic acid, ferulic acid and syringic acid in doxorubicin induced cardiotoxicity.
Materials-Methods: The rats were randomized into 5 equal groups each of six. Group 1 sham group, which received no treatment, group 2, received doxorubicin at a dose 3 mg/kg IP every other 2 days plus normal saline as vehicle orally and considered as Cardiotoxic Control, Group 3 received doxorubicin plus ferulic acid 10 mg/day p.o for 2 weeks, group 4 received doxorubicin plus caffeic acid 40 mg/day p.o for 2 weeks, group 5 received doxorubicin plus syringic acid 100 mg/day p.o for 2 weeks. Left venrricular function was measured by volume flow meter.

At the end of experiment and after recording the final body weight, blood samples were collected from the heart for measurement of plasma level of cardiac troponin I (cTn I) and serum level of oxidative stress parameter malondialdehyde (MDA) and also for measurement of high sensitive c-reactive protein (hs-CRP). The heart were weighted and then harvested. The apical side was fixed in 10% formalin for histological examination and the basal side was homogenized for the measurement of tissue tumor necrosis factor-α (TNF-α) and interleukin-1β (IL-1β) and interleukin-10 (IL-10).
Results: Compared with sham group, levels of myocardial TNF-α, IL-1βand IL-10; plasma cTnI were significantly increased ($p<0.001$) in Doxorubicin treated group with significant increase ($p<0.001$) in MDA, hs-CRP and significant impairment of left ventricular ejection fraction and cardiac out put ($p<0.001$). Caffeic acid, ferulic acid and syringic acid significantly counteracted the increase in myocardial TNF-alfa,IL-1-beta, IL-10, serum cTnI, hs-CRP and MDA. Additionally these compounds significantly (p,0.001) counteracted the decrease in ejection fraction and cardiac out put.
Conclusions: The results of the present study reveal that phenolic compounds (caffeic acid,ferulic acid and syringic acid have been shown to decrease doxorubicin-induced cardiotoxicity via interfering with inflammatory reactions and also prophylactic use of these compounds may ameliorate the left ventricular function which deteriorate by doxorubicin administration.

OP-132

The Cardioprotective Potential Of L-Carnitine in Myocardial Ischaemia/Reperfusion in Rats

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Background: Ischaemia /reperfusion induce myocyte necrosis and apoptosis that seem to be the prevalent mode of death during the ischemic period and the subsequent reperfusion. L-carnitine is essential for the transport and metabolism of fatty acids to the mitochondria for energy production.
Aim of the study: We aimed to study the possible cardio protective potential of L-carnitine in myocardial regional ischemia – reperfusion injury.
Materials-Methods: Dwale-sprague rats were divided into five groups (6 rats for each) assigned as I, II, III, IV, V. Group I (sham), rats were subjected for all surgical procedure without ligation of (LAD). GroupII (control), rats were subjected for entire surgical procedure with ligation of (LAD). GroupIII (control vehicle), rats pretreated with normal saline (vehicle for L-carnitine) for 7days then subjected for entire surgical procedure with ligation of (LAD). Group (L-carnitine treated group), rats previously treated with (100 mg/kg, IP) L-carnitine then subjected to entire surgical procedure including LAD ligation for 25 minutes followed by 120 minutes reperfusion. At the end of reperfusion,cardiac tissue TNF-α, IL-1β, IL-6 and ssDNA as well as plasma cardiac troponin I (cTnI) were measured.It has been found that L-carnitine treated group showed significant reduction ($\rho<0.05$) in TNFα, IL-1β, IL-6, ssDNA (a marker of apoptosis) and cTnI with respect to the control or control vehicle groups. Additionally histopathological study revealed that L-carnitine significantly ($\rho<0.05$) improved cardiac injury score as compared with control group. It is concluded from this study that L-carnitine reduce inflammatory reaction associated with ischemia/reperfusion and ameliorates apoptosis.

OP-133

Goserline Acetate Protects The Heart From Ischemia/Reperfusion Injury and Modulates Apoptosis in Rats

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Ischemia reperfusion and apoptosis both are serious complications of myocardial ischemia. Although the restoration of blood flow to an ischemic organ is essential to prevent irreversible tissue injury, reperfusion may augment's tissue injury in excess of that produced by ischemia alone. Restoration of blood flow to ischemic myocardium results in the ischemia reperfusion (I/R) injury.This study was undertaken to investigate the cardioprotective potential of goserline acetate in regional ischemia reperfusion and apoptosis of the male rat hearts.
Material-Method: Eighteen Swiss albino rats were randomized into 3 equal groups(6 in each group). Group 1 sham group, rat underwent the same anesthetic and surgical procedure as the control group except for surgical ligation of LAD, Group 2 control group, rats underwent surgical ligation of LAD and subjected to regional ischemia for 25 min and reperfusion for 40 min, Group 3 Goserelin acetate treated, rats underwent abdominal subcutaneous injection of Goserelin (LHRH analogue) 3wks before the surgery, then underwent surgical LAD ligation, and subjected to 25min of ischemia and 40min of reperfusion.At the end of experiment blood samples were collected from the heart for measurement of plasma level of cardiac troponin I(cTn I). The heart were harvested, and divided into 3 sections, the 1st for the measurement of cardiac apoptosis level, the 2nd homogenized for measurement of tissue (TNF-α,IL1-β,ICAM-1) and the 3rd was fixed in 10% formalin for histological examination.
Result: Compared with the sham group, levels of myocardial TNF-α & IL-1β, ICAM-1 and apoptosis; plasma cTn I were increased ($p<0.05$) in control group. Histologically, all control group rats showed significant myocardial injury ($p<0.05$) compared with sham group. Goserelin significantly counteract the increase in myocardial level of TNF-α, IL-1, ICAM-1, plasma cTn I and apoptosis ($p<0.05$). Histological analysis revealed that Goserelin markedly reduced ($p<0.05$) the severity of heart injury in the rats underwent the regional ischemia- reperfusion procedure.
Conclusion: Goserelin acetate may ameliorate regional I/R injury and apoptosis in the ischemic heart in rats via interfering with inflammatory pathways.

Congestive Heart Failure

OP-134

The Predictive Value of Intraventicular Dyssynchrony in Response to Levosimendan Therapy in Patients with Decompensated Heart Failure

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Purpose: We aimed to investigate the role of intraventricular dyssynchrony using echocardiography in predicting response to levosimendan therapy in patients with acute systolic heart failure.
Methods: Patients with an ejection fraction (EF) lower than 35% who required intravenous inotropic support despite optimal heart failure therapy were included to this study. Regional myocardial function was evaluated in eight segments (basal and middle segments of intraventricular septum, lateral, anterior, inferior walls) by tissue doppler imaging. Peak systolic velocities were measured for each segment. Interval